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EXAMINER	
ZARA, JANE J	

ART UNIT	PAPER NUMBER
1635	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/750,410	LI ET AL.	
	Examiner	Art Unit	
	Jane Zara	1635	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *14 April 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,15,16 and 18-22* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,15,16 and 18-22* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *4-14-08. 10-12-07*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

This Office action is in response to the communication filed 4-14-08.

Claims 1, 15, 16, and 18-22 are pending in the instant application.

Information Disclosure Statement

The listing of references in the Search Report is not considered to be an information disclosure statement (IDS) complying with 37 CFR 1.98. 37 CFR 1.98(a)(2) requires a legible copy of: (1) each foreign patent; (2) each publication or that portion which caused it to be listed; (3) for each cited pending U.S. application, the application specification including claims, and any drawing of the application, or that portion of the application which caused it to be listed including any claims directed to that portion, unless the cited pending U.S. application is stored in the Image File Wrapper (IFW) system; and (4) all other information, or that portion which caused it to be listed. In addition, each IDS must include a list of all patents, publications, applications, or other information submitted for consideration by the Office (see 37 CFR 1.98(a)(1) and (b)), and MPEP § 609.04(a), subsection I. states, "the list ... must be submitted on a separate paper." Therefore, the references cited in the Search Report have not been considered. Applicant is advised that the date of submission of any item of information or any missing element(s) will be the date of submission for purposes of determining compliance with the requirements based on the time of filing the IDS, including all "statement" requirements of 37 CFR 1.97(e). See MPEP § 609.05(a).

Response to Arguments and Amendments

Applicant's arguments with respect to claims 1, 15, 16, 18-22 have been considered but are moot in view of the new ground(s) of rejection set forth below. Arguments are addressed below that remain relevant to the new grounds of rejections, however.

Withdrawn Rejections

Any rejections not repeated in this Office action are hereby withdrawn.

Rejections Necessitated by Amendment

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1, 15, 16, and 18-22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Housman, Reeves et al, Anderson et al, Takiguchi et al and Milner et al, in view of Au-Young et al and Reed et al.

Housman (USPN 6,200,754) teaches nucleic acid constructs comprising an expression vector comprising a promoter operably linked to antisense and ribozymal oligonucleotides, which are complementary to a nucleic acid encoding a human DNA dependent protein kinase (DNA PK) subunit, which subunit is optionally Ku 70 or 80,

and which antisense or ribozyme is administered in a liposomal formulation to the target cells and inhibits the expression of the target DNA PK in vitro, and increases the sensitivity of a host cell comprising the antisense or ribozyme to DNA-damaging agents, including UV radiation (See esp. col. 27, 32, 41-42, 44, 52-53, claims 1, 3, 8, 10 and 11).

Reeves et al (J. Biol. Chem., Vol. 264(9): 5047-5052, 1989) teach the polynucleotide sequence encoding human DNA-PK subunit Ku70, and Ku70's binding to the ends of double stranded DNA in a complex with Ku80. Reeves also teaches the role of the Ku 70/80 complex in DNA repair and in autoimmunity (see esp. figure 4 on p. 5050 and the text on p. 5047, and text on p. 5052).

Anderson (TIBS, Vol. 18, pages 433-437, 1993) teaches the role of DNA-PK in coordinating nuclear processes and modulating checkpoint mechanisms activated by DNA damage (see text on pp. 433-434, fig. 1 on p. 435-437).

Takiguchi (Genomics 35: 129-135, 1996) teaches the role of mouse and human DNA-PK (comprising the subunits Ku70, Ku80 and DNA-PK catalytic subunit) in DNA repair, and an increase in a cell's sensitivity to DNA damaging agents with loss of DNA-PK function. Takiguchi teaches the DNA-dependent protein kinase (DNA-PK) comprising Ku70, Ku80 and the DNA-PK catalytic subunit, which DNA-PK functions in DNA double-strand break repair (see 1st & 3rd full paragraph on p. 129). Takiguchi also teaches that the Ku70/80 heterodimer portion of the DNA-PK binds with high affinity to the end of double stranded DNA, and to a single stranded DNA transition (see 2nd full paragraph on p. 129), and recruits and activates the DNA catalytic subunit of the DNA-

PK (see 3rd full paragraph on p. 129). Takiguchi teaches the role of Ku70 in DNA double strand repair (see bridging paragraph, pp. 133-134). Takiguchi teaches the motivation to establish experimental mouse models for studying the role of DNA-PK in human diseases (see last paragraph of the article, bridging pp. 133-134).

Milner (Nature Biotech. 15: 537-541, 1997) teaches methods of designing and testing antisense oligonucleotides for their ability to specifically hybridize and inhibit the expression of a target nucleic acid of known nucleotide sequence in vitro (See especially figures 5-7 on pages 539-540).

The primary references do not teach expression vectors comprising full length antisense or ribozymes, nor vectors comprising operably linked inducible promoters such as the heat shock promoter in combination with liposomes for adminstration to target cells.

Au-Young (USPN 5,773,580) teaches pharmaceutical compositions comprising antisense oligonucleotides for inhibiting a known target gene, as well as teaching expression vectors comprising antisense oligonucleotides and ribozymes, which oligonucleotides are operably linked to regulatory elements including an inducible (heat shock) promoter, and administration of these nucleic acid constructs in combination with liposomes (see esp. col. 10-11, 20-21).

Reed et al (Proc. Natl. Acad. Sci., Vol. 87, pages 3660-3664, 1990) teach full length antisense in appropriate expression vectors, operably linked to a promoter, for transfecting target cells and inhibiting the expression of a target gene of interest (see

esp. second paragraph of the methods section on p. 3660, fig. 1 on p. 3661, fig. 4 on p. 3663).

The claims are drawn to expression vectors comprising antisense or ribozymal oligonucleotides complementary to a nucleic acid encoding a human DNA dependent protein kinase subunit, which subunit is optionally Ku 70 or 80, which antisense or ribozyme inhibits the expression of the target DNA PK subunit in a target host cell in vitro, and which antisense or ribozyme is operably linked, in an appropriate expression vector, to a heat shock promoter, and which target cell comprising the antisense or ribozyme has increased sensitivity to a DNA damaging agent, including radiation induced DNA damage.

It would have been obvious to one of ordinary skill in the art to design and utilize antisense oligonucleotides to inhibit the expression of a human DNA dependent protein kinase subunits including Ku 70 and 80, both of known sequences, in vitro, because the sequences of the subunits for human DNA dependent protein kinase had been taught previously by Reeves et al and Housman, and Milner et al teach the ability to design and assess antisense oligonucleotides for their ability to inhibit the expression of a target gene of known nucleotide sequence in vitro using routine screening assays that are well known in the art (see Milner at pages 539-540). It would have been obvious to one of ordinary skill in the art to inhibit the expression of the known sequences encoding the subunits of human DNA dependent protein kinase of known nucleotide sequence in vitro using antisense oligonucleotides because the methods for inhibiting a target gene of known sequence using antisense had been taught previously by Milner et al and such

methods of screening antisense in vitro for inhibition of target gene expression were routine at the time the invention was made. Milner et al additionally teach methods of designing and evaluating antisense which target different regions of a target gene of previously disclosed sequence for their ability to inhibit a target gene in vitro.

One of ordinary skill in the art would have expected that the methods of designing and assessing antisense oligonucleotides for inhibiting a target gene of known sequence, which were taught by Milner et al, to be routine for a previously characterized target gene, would successfully be used to identify numerous antisense oligonucleotides human DNA dependent protein kinase subunits, including KU70. It would have also been obvious to utilize full length antisense for target gene inhibition because this had been done previously by many in the art, including Reed, for inhibiting the expression of a known target gene.

It would have been obvious to one of ordinary skill in the art to insert antisense oligonucleotides or full length antisense constructs into an appropriate expression vector, operably linked to an inducible promoter including a heat shock promoter, and administer to a target cell in liposomal formulations, because such expression systems have been used routinely in the art for expression of nucleic acid constructs including antisense and ribozymes in an appropriate target cell, as taught previously by Au-Young et al, and liposomes were well known in the art to enhance target cell uptake. One of ordinary skill in the art would have been motivated to operably link an antisense oligonucleotide to an inducible promoter in an appropriate expression vector in order to control the conditions of expression of the operably linked antisense, and in order to

control conditions for antisense expression and subsequent inhibition of the antisense's target gene in an appropriate target cell. One of ordinary skill in the art would have been motivated to target and inhibit the expression of DNA-PK in order to increase a target cell's sensitivity to DNA damaging agents (e.g. a target cancer cell), because Takiguchi et al teach the relationship between increasing cell radiosensitivity or loss of DNA repair function, and loss of functional DNA-PK.

One of ordinary skill in the art would have expected that by utilizing appropriate conditions for expression (e.g. induction by heat), the antisense targeting DNA-PK would be expressed upon induction of the heat shock promoter because such induction systems as heat shock promoters have been routinely used as described by Au-Young et al. One of ordinary skill in the art would have been motivated to induce expression of antisense and ribozymes under desired conditions (e.g. upon exposure to heat, delivered to a cell in a liposomal formulation to enhance cellular uptake and reduce nucleic acid degradation) because induction is a way of controlling the conditions for increased expression of the operably linked antisense and ribozymes, and also a way of controlling the subsequent inhibition of target gene expression following expression of these antisense. In this way, increasing a cell's sensitivity to DNA damaging agents is in turn induced following heat treatment and expression of antisense. Therefore the invention as a whole would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made.

It would have been obvious to one of ordinary skill in the art to design and utilize antisense oligonucleotides to inhibit the expression of Ku70 in vitro because its

nucleotide sequence had been taught previously by Reeves et al, and Milner et al teach the ability to design and assess antisense oligonucleotides for their ability to inhibit the expression of a target gene of known nucleotide sequence in vitro using routine screening assays that are well known in the art (see Milner at pages 539-540). Milner et al additionally teach methods of designing and evaluating antisense which target different regions of a target gene of previously disclosed sequence for their ability to inhibit a target gene in vitro. One of ordinary skill in the art would have expected that the methods of designing and assessing antisense oligonucleotides for inhibiting a target gene of known sequence, which were taught by Milner et al, to be routine for a previously characterized target gene, would successfully be used to identify numerous antisense oligonucleotides human DNA dependent protein kinase subunits, including Ku70.

One of ordinary skill in the art would have been motivated to target and inhibit the expression of the various subunits of DNA-PK, including Ku70, in order to increase a target cell's sensitivity to DNA damaging agents because Takiguchi et al teach the relationship between increasing cell radiosensitivity or loss of DNA repair function, and loss of functional DNA-PK. One of ordinary skill in the art would have been motivated to inhibit the expression of Ku70 in order to increase a target cell's sensitivity to DNA repair because it was well known in the art that Ku 70 is involved in double stranded DNA repair and it was also well known that strand repair occurs in cells following DNA damage (e.g. strand breaks). One of ordinary skill in the art would have expected that a cancer cell would undergo DNA repair after its exposure to DNA damaging agents.

And one of ordinary skill in the art would be motivated to undermine a cancer cell's ability to repair DNA after treating it with DNA damaging agents in order to eventually undermine that cancer cell's ability to survive.

One of ordinary skill in the art would have been motivated to inhibit the expression of various subunits of DNA PK in vitro in order to study their role(s) in various cellular processes because Anderson teaches the purported role of DNA-PK in coordinating nuclear processes and modulating checkpoint mechanisms activated by DNA damage

Takiguchi teaches Ku70's role in DNA double stranded break repair. Takiguchi teaches the DNA-dependent protein kinase (DNA-PK) comprising Ku70, Ku80 and the DNA-PK catalytic subunit, which DNA-PK functions in DNA double-strand break repair (see 1st & 3rd full paragraph on p. 129). Takiguchi also teaches that the Ku70/80 heterodimer portion of the DNA-PK binds with high affinity to the end of double stranded DNA, and to a single stranded DNA transition (see 2nd full paragraph on p. 129), recruits and activates the DNA catalytic subunit of the DNA-PK (see 3rd full paragraph on p. 129). Takiguchi teaches the role of Ku70 in DNA double strand repair (see bridging paragraph, pp. 133-134). The role of Ku70 in DNA double strand breaks repair was not a surprising finding at the time the instant application was filed.

Takiguchi teach the role of mouse and human DNA-PK in DNA repair, and an increase in a cell's sensitivity to DNA damaging agents with loss of DNA-PK function. Takiguchi teaches the DNA-dependent protein kinase (DNA-PK) comprising Ku70, Ku80 and the DNA-PK catalytic subunit, which DNA-PK functions in DNA double-strand

break repair (see 1st & 3rd full paragraph on p. 129). Takiguchi also teaches that the Ku70/80 heterodimer portion of the DNA-PK binds with high affinity to the end of double stranded DNA, and to a single stranded DNA transition (see 2nd full paragraph on p. 129), and recruits and activates the DNA catalytic subunit of the DNA-PK (see 3rd full paragraph on p. 129). Takiguchi teaches the role of Ku70 in DNA double strand repair (see bridging paragraph, pp. 133-134). Takiguchi teaches the motivation to establish experimental mouse models for studying the role of DNA-PK in human diseases (see last paragraph of the article, bridging pp. 133-134). Takiguchi therefore teaches a motivation to inhibit either mouse DNA-PK or human Ku70 in DNA-PK activity to study its role in human diseases, including the ability of a cell with DNA damage to repair strand breaks. This motivation, combined with the routine approach taught by Milner to design and test antisense in their ability to target and inhibit the expression of a target gene of known sequence (e.g. human Ku70) in vitro, renders the invention obvious.

Takiguchi provides the motivation to target and inhibit the expression of Ku70 in humans (or in a mouse model) to study its role in various human diseases, and to study the role of Ku70 in DNA-PK's ability to repair strand breaks. It was well known in the art that when damaging agents are used to treat cells, strand breaks occur. Milner taught the routine approach to design and test antisense inhibition in a cell in vitro and Reeves teaches the nucleotide sequence of the target gene. It therefore would have been obvious to one of ordinary skill in the art to design antisense to inhibit the expression of the well known target gene Ku70 in vitro to study its role in repairing DNA strand breaks. It would have been obvious to compare the effect of DNA damaging agents on cells that

have Ku70 expression with cells that lack Ku70 expression following the inhibition by antisense. For these reasons, the combined teachings of Reeves et al, Anderson et al, Takiguchi et al and Milner et al, Au-Young et al and Reed et al render the instant invention obvious.

Applicant argues that Reeves does not teach full length antisense that target human Ku70 and that the remaining references cited in the instant rejection do not cure the deficiency because Reed, for instance, teaches full length antisense that target BCL-2, which is a different target gene. Contrary to Applicant's assertions, constructing antisense polynucleotides that target a specific gene of interest and testing them for their ability to inhibit target gene expression was well known and involved routine experimentation at the time of the instant invention. Since the instant references have been cited for obviousness, not for anticipation, combining the teachings of Reed, who used a full length antisense to inhibit expression of a well known target gene, with those references disclosing the target sequences of Ku70 and 80, as well as the motivation to inhibit Ku70 and Ku80, properly render the instant invention obvious.

Applicant also argues that Agrawal teaches the unpredictability of antisense, and Milner discusses the variable successs commonly experienced in choosing antisense oligonucleotides for target gene inhibition, supporting Applicant's reasoning that the instant invention is not obvious. Contrary to Applicant's assertions, the nucleic acid sequences of the instantly claimed target genes were well known in the art at the time the instant invention was made, the methods for designing and testing antisense for target gene inhibition were also well known in the art at the time of filing. So, utilizing

this knowledge, and utilizing these well known techniques, would have involved routine experimentation to determine whether target gene inhibition occurs. One of skill in the art would have had a reasonable expectation of success in finding antisense that would inhibit the well known target genes, Ku70 and 80, in vitro. Although one might not have known before testing their ability to inhibit target gene expression which antisense were optimal, testing such antisense, including full length antisense, for their ability to inhibit target gene expression in vitro would have involved routine experimentation. Therefore, the instant obviousness rejection is maintained.

Applicant additionally argues that Reeves does not teach or suggest in vitro liposome administration of antisense oligonucleotides, and therefore the instant invention would not be obvious over the combined references. Contrary to Applicant's assertions, liposomes were well known in the art at the time of the instant invention for enhancing delivery of nucleic acids to target cells.

See, for example, the following excerpted text from USPN 6,200,754 (Housman et al):

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Agents intended to be administered intracellularly may be administered using techniques well known to those of ordinary skill in the art. For example, such agents may be encapsulated into liposomes, then administered as described above. Liposomes are spherical lipid bilayers with aqueous interiors. All molecules present in an aqueous solution at the time of liposome formation are incorporated into the aqueous interior. The liposomal contents are both protected from the external microenvironment and, because liposomes fuse with cell membranes, are efficiently delivered into the cell cytoplasm. Additionally, due to their hydrophobicity, small organic molecules may be directly administered intracellularly.

In addition, USPN 5,773,580 (Au-Young et al) also teaches the routine use of liposomal formulations in delivery of nucleic acids to target cells, as the following excepted text illustrates:

> "Delivery by transfection and by liposome are quite well known in the art."

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1, 15, 16 and 18-24 are provisionally rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 27, 39 and 40 of copending Application No. 10/712,642. Although the conflicting claims are not identical, they are not patentably distinct from each other because claims 1, 15, 16 and 18-24 are drawn to compositions and methods for increasing a cell's susceptibility to DNA damaging agents comprising administration of antisense that target and inhibit

expression of Ku70 and claims 27 and 28 of Application No. 10/712,642 are drawn to compositions and methods for increasing a cell's susceptibility to DNA damaging agents comprising administration of an expression vector comprising antisense that target and inhibit expression of Ku70.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

No arguments have been made addressing this rejection.

Conclusion

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Certain papers related to this application may be submitted to Art Unit 1635 by facsimile transmission. The faxing of such papers must conform with the notices published in the Official Gazette, 1156 OG 61 (November 16, 1993) and 1157 OG 94 (December 28, 1993) (see 37 C.F.R. ' 1.6(d)). The official fax telephone number for the

Group is 571-273-8300. NOTE: If Applicant does submit a paper by fax, the original signed copy should be retained by applicant or applicant's representative. NO DUPLICATE COPIES SHOULD BE SUBMITTED so as to avoid the processing of duplicate papers in the Office.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Jane Zara whose telephone number is (571) 272-0765. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James Douglas Schultz, can be reached on (571) 272-0763. Any inquiry of a general nature or relating to the status of this application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Jane Zara
6-30-08

/Jane Zara/
Primary Examiner, Art Unit 1635